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     (FILE 'HOME' ENTERED AT 09:25:59 ON 01 NOV 2003)

     FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, BIOSIS, BIOTECHNO, CANCERLIT,
     CAPLUS, CEN, DISSABS, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL,
     DRUGU, EMBAL, EMBASE, ESBIOBASE, IFIPAT, IPA, JICST-EPLUS, KOSMET,
     LIFESCI, MEDICONF, MEDLINE, NAPRALERT, NLDB, NUTRACEUT, ..' ENTERED AT
     09:26:12 ON 01 NOV 2003
L1          747 S (RECESSIVE DYSTROPHIC EPIDERMOLYSIS BULLOSA)/AB
L2          410 S (RECESSIVE DYSTROPHIC EPIDERMOLYSIS BULLOSA)/TI AND L1
L3          424 S (RECESSIVE AND DYSTROPHIC AND EPIDERMOLYSIS AND BULLOSA)/TI A
L4         1233 S (RECESSIVE AND DYSTROPHIC AND EPIDERMOLYSIS AND BULLOSA)/TI
L5          456 DUP REM L4 (777 DUPLICATES REMOVED)
L6            1 S L5 AND (BASIC AND FIBROBLAST AND GROWTH)/TI
L7            0 S L6 AND (CURCUMIN OR DEMETHOXYCURCUMIN)

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L9 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN

SO Journal of Pharmacy and Pharmacology, (**1997**) Vol. 49, No. 1, pp. 105-107.
CODEN: JPPMAB. ISSN: 0022-3573.

AB Because **curcumin**, a compound with anti-inflammatory and anticancer activity, inhibits induction of nitric oxide synthase in activated macrophages and has been shown to be a potent scavenger of free radicals we have investigated whether it can scavenge nitric oxide directly. **Curcumin** reduced the amount of nitrite formed by the reaction between oxygen and nitric oxide generated from sodium nitroprusside. Other related compounds, e.g. **demethoxycurcumin**, bisdemethoxycurcumin and diacetylcurcumin were as active as **curcumin**, indicating that the methoxy and the phenolic groups are not essential for the scavenging activity. The results indicate **curcumin** to be a scavenger of nitric oxide. Because this compound is implicated in inflammation and **cancer**, the therapeutic properties of **curcumin** against these conditions might be at least partly explained by its free-radical scavenging properties, including those toward nitric oxide.

IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics); Pharmacology; Tumor Biology

IT Chemicals & Biochemicals
NITRIC OXIDE; **CURCUMIN**; NITRIC OXIDE SYNTHASE; **DEMETHOXYCURCUMIN**; DIACETYLCURCUMIN

IT Miscellaneous Descriptors
ANTIINFLAMMATORY AGENT; ANTINEOPLASTIC AGENT; BISDEMETHOXYCURCUMIN; **CURCUMIN**; **DEMETHOXYCURCUMIN**; DIACETYLCURCUMIN; NITRIC OXIDE; NITRIC OXIDE SYNTHASE; PHARMACOLOGY

RN 10102-43-9 (NITRIC OXIDE)
458-37-7 (**CURCUMIN**)
125978-95-2 (NITRIC OXIDE SYNTHASE)
22608-11-3 (**DEMETHOXYCURCUMIN**)
19697-86-0 (DIACETYLCURCUMIN)

L4 ANSWER 13 OF 33 CAPLUS COPYRIGHT 2003 ACS on STN

TI A collagen XVIII fragment as an inhibitor of **angiogenesis** and its therapeutic uses in the treatment of **angiogenesis**-dependent cancers

PI WO 9715666 A1 **19970501**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 9715666	A1	19970501	WO 1996-US16925	19961023	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI					
US 5854205	A	19981229	US 1996-740168	19961022	<--
AU 9674666	A1	19970515	AU 1996-74666	19961023	<--
AU 717277	B2	20000323			
EP 857210	A1	19980812	EP 1996-936842	19961023	<--
EP 857210	B1	20030903			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
CN 1202932	A	19981223	CN 1996-198480	19961023	<--
BR 9611174	A	19990914	BR 1996-11174	19961023	
JP 2002501362	T2	20020115	JP 1997-516724	19961023	
AT 248912	E	20030915	AT 1996-936842	19961023	
NO 9801803	A	19980617	NO 1998-1803	19980422	<--
US 2002086352	A1	20020704	US 1998-174282	19981016	
US 6544758	B2	20030408			
US 2002127595	A1	20020912	US 1998-174516	19981016	

AB Endostatin, an inhibitor of endothelial cell proliferation that is capable of inhibiting **angiogenesis** and causing tumor regression is described for therapeutic use. The protein has potential therapeutic use in a no. disease assocd. with abnormal **angiogenesis**. Endostatin is approx. 20 kDa and corresponds to a C-terminal fragment of collagen type XVIII, and methods of treating **angiogenesis**-related disease. The protein was identified in conditioned medium from the **hemangioendothelioma** cell line EOMA using inhibition of proliferation of cultured endothelial cells. Inhibition was specific to endothelial cells. The protein was. . .

L4 ANSWER 2 OF 33 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:274115 BIOSIS
DN PREV199800274115
TI Antiangiogenesis and apoptosis as mediators of concomitant tumor resistance induced by Calu-6, a human lung carcinoma cell line, in nude mice.
AU Bonfil, R. Daniel [Reprint author]; Bustuoabad, Oscar D.; Binda, M. Mercedes
CS Lab. Fundacion Invest. Cancer CEFYBO, Serrano 669, 1414 Buenos Aires, Argentina
SO Oncology Research, (**1998**) Vol. 10, No. 1, pp. 15-21. print.
CODEN: ONREE8. ISSN: 0965-0407.
DT Article
LA English
ED Entered STN: 24 Jun 1998
Last Updated on STN: 24 Jun 1998
AB Concomitant resistance (CR), the phenomenon by which tumor-bearing hosts are able to inhibit secondary implants of the same tumor at distant sites of the body, has been previously observed by us and others in different murine tumor models. Here, we verified the generation of CR in nude mice by tumors induced by SC inoculation of Calu-6, a human lung carcinoma cell line. Histological analysis of secondary tumors subject to CR did not reveal macrophage infiltration nor cytotoxic signs. Although serum from tumor-bearing mice inhibited in vitro (3H)thymidine uptake by Calu-6 cells, no significant differences in (3H)thymidine labeling index of tumors implanted in the right flank of mice with and without a primary tumor in the left flank were detected. In our model, the presence of a primary tumor hindered remote tumor **angiogenesis**, as well as serum from tumor-bearing mice inhibited in vitro proliferation of an endothelial cell line derived from a murine **hemangioendothelioma** . Conversely, an enhancement of the apoptotic index was observed in secondary tumor implants carried out in tumor-bearing mice. The results reported herein show that human tumor cells are capable of inducing CR, and that this phenomenon would be a consequence of an impaired neovascularization as well as an increased programmed cell death at sites distant from the primary tumor.
CC Neoplasms - Pathology, clinical aspects and systemic effects 24004
Cytology - Human 02508
Respiratory system - Pathology 16006
IT Major Concepts
Tumor Biology
IT Miscellaneous Descriptors
antiangiogenesis; apoptosis; tumor growth; tumor resistance
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Calu-6: human lung carcinoma
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
nude mouse
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

L4 ANSWER 3 OF 33 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1997:511206 BIOSIS

DN PREV199799810409
TI Tenascin-C expression in ultrastructurally defined angiogenic and vasculogenic lesions.
AU Kostianovsky, Mery [Reprint author]; Greco, M. Alba; Cangiarella, Joan; Zagzag, David
CS Dep. Pathol., Anat. Cell Biol., Thomas Jefferson Univ., 1015 Chestnut, Suite 902, Philadelphia, PA 19107, USA
SO Ultrastructural Pathology, (**1997**) Vol. 21, No. 6, pp. 537-544.
CODEN: ULPAD3. ISSN: 0191-3123.
DT Article
LA English
ED Entered STN: 10 Dec 1997
Last Updated on STN: 10 Dec 1997
AB Tenascin-C (TN) is an extracellular matrix glycoprotein expressed during embryogenesis. Its distribution is restricted in normal adult tissues and is upregulated in tumors and inflammatory conditions. Twenty-five specimens were studied, including 7 reactive vascular lesions (6 cases of granulation tissue and 1 case of bacillary angiomatosis), and 18 vascular tumors (6 angiosarcomas, 7 hemangioendotheliomas, and 5 AIDS-related nodular type Kaposi's sarcomas). Formalin fixed-paraffin-embedded tissues were stained with monoclonal antibody to TN (DAKO) and with MIB-1 (AMAC). Heterogeneous expression of TN immunoreactivity was seen in all cases, with a diffuse pattern in bacillary angiomatosis and most granulation tissue cases and a focal pattern in angiosarcoma and most **hemangioendothelioma** cases. Kaposi's sarcoma cases showed both a focal and diffuse pattern of distribution. In most cases proliferation indices (PI) did not correlate with TN expression. Electron microscopy demonstrated active **angiogenesis** in bacillary angiomatosis and granulation tissue and vasculogenesis in angiosarcoma and **hemangioendothelioma**. The study demonstrated positive TN expression in reactive lesions with **angiogenesis** (granulation tissue and bacillary angiomatosis) and neoplastic lesions showing vasculogenesis (angiosarcoma and **hemangioendothelioma**), although with a different pattern of distribution. These results suggest that TN might be an important extracellular matrix glycoprotein in **angiogenesis** and vasculogenesis.
CC Microscopy - Electron microscopy 01058
Cytology - Human 02508
Biochemistry studies - Proteins, peptides and amino acids 10064
Biophysics - Methods and techniques 10504
Biophysics - Molecular properties and macromolecules 10506
Anatomy and Histology - Microscopic and ultramicroscopic anatomy 11108
Cardiovascular system - Blood vessel pathology 14508
Neoplasms - Pathology, clinical aspects and systemic effects 24004
IT Major Concepts
Biochemistry and Molecular Biophysics; Cardiovascular Medicine (Human Medicine, Medical Sciences); Cell Biology; Methods and Techniques; Morphology; Oncology (Human Medicine, Medical Sciences)
IT Miscellaneous Descriptors
ANGIOGENIC LESIONS; ANGIOSARCOMA; CARDIOVASCULAR SYSTEM; ELECTRON MICROSCOPY; EMBRYOGENESIS; EXPRESSION; EXTRACELLULAR MATRIX GLYCOPROTEIN; **HEMANGIOENDOTHELIOMA**; KAPOSI'S SARCOMA; MICROSCOPY METHOD; NEOPLASTIC DISEASE; TENASCIN-C; ULTRASTRUCTURALLY DEFINED; VASCULAR LESIONS; VASCULOGENESIS
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

L25 ANSWER 6 OF 26 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
TI The prognostic significance of **basic fibroblast growth factor** in cutaneous **malignant melanoma**.
SO Journal of Cutaneous Pathology, (**1996**) Vol. 23, No. 6, pp. 506-510.
CODEN: JCUPBN. ISSN: 0303-6987.
AB **Basic fibroblast growth factor** (bFGF) is a growth factor and an **angiogenesis** factor which may play a role in the evolution of cutaneous **malignant melanoma** (CMM). In this study, we evaluated the distribution of bFGF in CMM using immunochemical methods and correlated the pattern of. .
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L19 ANSWER 2 OF 25 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:321336 BIOSIS
DN PREV199800321336
TI Vascular endothelial growth factor and basic fibroblast growth factor present in Kaposi's sarcoma (KS) are induced by inflammatory cytokines and synergize to promote vascular permeability and KS lesion development.
AU Samaniego, Felipe; Markham, Phillip D.; Gendelman, Rita; Watanabe, Yoshiki; Kao, Vivien; Kowalski, Kimberly; Sonnabend, Joseph A.; Pintus, Aldo; Gallo, Robert C.; Ensoli, Barbara
CS Lab. Virol., Ist. Superiore di Sanita, Viale Regina Elena 299, 00161 Rome, Italy
SO American Journal of Pathology, (**June, 1998**) Vol. 152, No. 6, pp. 1433-1443. print.
CODEN: AJPAA4. ISSN: 0002-9440.
DT Article
LA English
ED Entered STN: 22 Jul 1998
Last Updated on STN: 22 Jul 1998
TI Vascular endothelial growth factor and basic fibroblast growth factor present in Kaposi's sarcoma (KS) are induced by inflammatory cytokines and synergize to promote vascular permeability and KS lesion development.
AB All forms of **Kaposi's sarcoma** (KS) are characterized by spindle cell proliferation, **angiogenesis**, inflammatory cell infiltration, and edema. We have previously reported that spindle cells of primary KS lesions and KS-derived spindle cell cultures express high levels of **basic fibroblast** growth factor (bFGF), which is promoted by the inflammatory cytokines identified in these lesions. These cytokines, namely, tumor necrosis factor, interleukin-1, and Interferon-gamma, induce production and release of bFGF, which stimulates **angiogenesis** and spindle cell growth in an autocrine fashion. Here we show that both AIDS-KS and classical KS lesions co-express vascular endothelial growth factor (VEGF) and bFGF. VEGF production by KS cells is promoted synergistically by inflammatory cytokines present in conditioned media from activated T cells and in KS lesions. KS cells show synthesis of VEGF isoforms that are mitogenic to endothelial cells but not to KS spindle cells, suggesting a prevailing paracrine effect of this cytokine. This may be due to the level of expression of the flt-1-VEGF receptor that is down-regulated in KS cells as compared with endothelial cells. KS-derived bFGF and VEGF synergize in inducing endothelial cell growth as shown by studies using both neutralizing antibodies and antisense oligodeoxynucleotides directed against these cytokines. In addition, VEGF and bFGF synergize to induce angiogenic KS-like lesions in nude mice and vascular permeability and edema in guinea pigs. These results indicate that inflammatory cytokines present in KS lesions stimulate the production of bFGF and VEGF, which, in turn, cooperate to induce **angiogenesis**, edema, and KS lesion formation.